Infectious and Parasitic Diseases of the Gray Wolf and Their Potential Effects on Wolf Populations in North America

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Numerous infectious and parasitic diseases have been reported for the gray wolf, including more than 10 viral, bacterial, and mycotic diseases and more than 70 species of helminths and ectoparasites. However, few studies have documented the role of diseases in population dynamics. Disease can affect wolf populations directly by causing mortality or indirectly by affecting physiological and homeostatic processes, thriftiness, reproduction, behavior, or social structure. In addition, wolves are hosts to diseases that can affect prey species, thus affecting wolf populations indirectly by reducing prey abundance or increasing vulnerability to predation. Diseases such as canine distemper and infectious canine hepatitis are enzootic in wolf populations, whereas rabies occurs in wolves primarily as a result of transmission from other species such as arctic and red foxes. Contact between wolves and domestic pets and livestock may affect the composition of diseases in wolves and their effects on wolf populations. Dogs were suspected of introducing lice and canine parvovirus to several wolf populations. The latter disease appears to have had initial demographic effects and is now enzootic in several wolf populations. The potential for diseases to affect wolf populations and other wild and domestic animals should be considered in wolf management plans, particularly in plans for reintroduction of wolves to areas within their former range.

Introduction

Published information on infectious and parasitic diseases of the gray wolf (*Canis lupus*) is largely composed of case reports, parasite surveys, and serological surveys of viral and bacterial pathogens. The effects of diseases on wolf population dynamics are largely unstudied, with the exception of scattered descriptive accounts of epizootics (Rausch 1958, Chapman 1978, Todd et al. 1981, Carbyn 1982b). Knowledge of the actual and potential influence of diseases on wolf populations can be important to wolf management programs, particularly for endangered populations and in areas where wolves are reintroduced. We review current literature on infectious and parasitic diseases in wolves, summarize information on the role of diseases in wolf population dynamics, and discuss considerations of disease in management of wolves.

Methods

We searched data bases for publications about parasites and diseases of wolves in *Current Contents, Wildlife Abstracts, Wildlife Review, Biosis*, and *Biological Abstracts*. Additional information was obtained from personal communication with researchers of diseases of wolves and other canids. We review these sources of information to provide an overview of the occurrence and significance of diseases of gray wolves in North America. Reference is made to diseases in other canid species when little or no information was available for the wolf, but when the disease is of potential significance to wolves.

Viral Diseases

Rabies

Rabies has probably occurred sporadically in nearly all, if not all, areas wolves have occupied (e.g., Cowan 1949, Mech 1970, Rausch 1973, Tabel et al. 1974, Custer and Pence 1981a, Sidorov et al. 1983, Butzeck 1987, Zarnke and Ballard 1987, Theberge et al. 1994). Mech (1970) identified rabies as one of the most important diseases of wild wolves, and Murie (1944) and Cowan (1949) speculated that rabies might limit wolf numbers. Since the early 1970's, several accounts have suggested that rabies may be an important

periodic or local cause of mortality. However, documented accounts of rabies in North American wolves are few, and the role of rabies in population regulation is unknown.

Ironically, as widespread as rabies is, relatively little information exists beyond the effects on individual wolves. Rausch (1973) reported that wolves without rabies appear to avoid individuals with the disease, but supporting evidence was not provided. Infected wolves may attack other wolves. Chapman (1978) reported the occurrence of rabies in one wolf pack on the Brooks Mountain Range in Alaska during 1977. He observed one wolf, which was later diagnosed with rabies, actively attacking other pack members. Subsequently, at least seven of 10 pack members died within five days, and rabies was confirmed in three wolves tested. At least five of the wolves died at two rendezvous sites, causing Chapman (1978) to speculate that rabid wolves tend to seek or remain in familiar areas, and therefore are not likely to transmit the disease to other wolf packs.

Rabies was one of several factors suspected to have contributed to a major decline of wolves in northwest Alaska after 1976 (Davis et al. 1980). However, only one wolf from the area was actually confirmed with rabies (Ritter 1981). Theberge et al. (1994) reported that rabies accounted for 21% of mortality among 29 radio-collared wolves that died in Algonquin Provincial Park, Ontario, during 1987-1992. Mortality from rabies occurred in three different packs within a nine-month period.

Wolves are not considered the primary vector of rabies, except in several countries in the eastern Mediterranean (Sikes 1970) and Asia (MacDonald and Voigt 1985). Wolves usually contract the disease from other vector species such as arctic foxes (*Alopex lagopus*) and red foxes (*Vulpes vulpes*) (Mech 1970, Rausch 1973, Ritter 1981, Theberge et al. 1994). A rabies epizootic among arctic and red foxes occurred in northwest Alaska (D. Ritter, Alaska Public Health Laboratory, Univ. of Alaska, pers. commun.). During February and late April 1990, three radio-collared wolves from two packs were found dead, and were diagnosed as having died from rabies (W.B. Ballard, Cooperative Wildlife Research Unit, Faculty of Forestry, University of New Brunswick, unpubl. data). During the ensuing three months, an additional five radio-collared wolves were found dead. Decomposition of the carcasses precluded testing for rabies, but this disease was suspected because no physical injuries were evident (Ballard et al. 1990). By 1 August 1990, eight radio-collared wolves in four packs had died from rabies (five of 21, one of seven, one of two, and one of one) (W.B. Ballard, unpubl. data). For rabies to have been transmitted between packs, infected wolves either had contact with adjacent pack members along territory edges, or dispersed into other pack areas. All known wolf deaths occurred within the known territory of each pack. Wolves in this area are generally not migratory, although packs may follow migrating caribou to their wintering grounds in some years (Ballard et al. 1990). Although dispersal could account for transmission of the disease in some cases, it is most likely that arctic and red foxes spread the disease to wolves. The rabies epizootic apparently did not spread beyond the four packs. Thus, rabies can eliminate single wolf packs and at times be a significant cause of mortality in a wolf population.

Canine distemper

Canine distemper has been reported in captive wolves since 1904 (Budd 1981), but Choquette and Kuyt (1974) were apparently the first to demonstrate serological evidence of infection in wild wolves in northern Canada (two of 86 seropositive). Because the wolf population had been substantially reduced prior to their study, they suggested that the low prevalence of distemper in the Northwest Territories may have been due to lowered opportunity for exposure in the reduced population. They suggested, based on the work of Trainer and Knowlton (1968) on coyotes (*Canis latrans*), that distemper was enzootic in wolves and only became an important mortality factor when compounded by other factors; e.g., crowding and malnutrition.

Stephenson et al. (1982) reported that wolves in three areas of Alaska (Nelchina Basin, Tanana Flats, and Yakutat) were seropositive for distemper. The relatively low seroprevalence (6–12%) suggested that exposure was either rare or perhaps fatal. Zarnke and Ballard (1987) further examined the wolf population in the Nelchina Basin for exposure to distemper during 1975 through 1982 (12% seroprevalence). Seropositive wolves were present during six of eight years, suggesting that distemper was enzootic in this population. Zarnke and Ballard (1987) also compared frequency of exposure to distemper in the wolf population to the frequency of the disease in dogs (*Canis familiaris*) from the area and concluded that dogs were not a direct source of infection for wolves. No deaths were attributed to distemper in any of 150 radio-collared wolves. In north-central Minnesota, 48% of 71 wolves sampled from 1977 to 1984 were seropositive for distemper (T.K. Fuller, Univ. of Massachusetts, pers. commun.).

Carbyn (1982b) was the first to provide evidence that distemper caused mortality in free-ranging wolves. Three of five known deaths from disease in Riding Mountain National Park, Manitoba, were caused by distemper, and he suggested that the number discovered was far less than the actual number. All known deaths occurred in five- to eight-month-old pups. Carbyn (1982b) concluded that diseases contributed to the 50% decline in the wolf population in the park. Distemper was the second largest known mortality factor. Peterson et al. (1984) also reported deaths of two yearling wolves from distemper in 1978 and 1980 on the Kenai Peninsula, Alaska.

Other than the accounts provided by Carbyn (1982b) and Peterson et al. (1984), there is no evidence that distemper is a significant mortality factor in wolves. Distemper usually infects dog pups at three to nine weeks of age (Gillespie and

Carmichael 1968), so mortality from distemper in wolves could easily occur undetected. However, most wolf populations in North America exhibit good recruitment, therefore distemper is not likely an important source of mortality.

Infectious canine hepatitis

Choquette and Kuyt (1974) were the first to report the serological evidence for infectious canine hepatitis (ICH) in free-ranging wolves in northern Canada: 11 (13%) of 86 wolves tested were seropositive. Stephenson et al. (1982) reported 100% exposure to ICH in three wolf populations in the Tanana Flats and Nelchina Basins, Alaska, during 1976-1979, but only a 40% exposure in northwest Alaska. Zarnke and Ballard (1987) reported an overall antibody prevalence of 81% in the Nelchina wolf population during 1975 through 1982. Annual prevalence varied from 72 to 100%. Both studies concluded that exposure to ICH was much higher in Alaska than in northern Canada. Forty-two percent of the exposed wolves were pups, suggesting early exposure. Zarnke and Ballard (1987) concluded that ICH was enzootic in Alaskan wolves. There was no relation between the occurrence of the disease in domestic dogs and seroprevalence in free-ranging wolves. Mortality in wolves from ICH has not been reported.

Canine parvovirus

Canine parvovirus (properly designated CPV-2) is a relatively new infectious organism that appeared in 1976 or 1977 in Europe and was first recognized as a disease agent in dogs in 1978 (Pollock 1984). CPV-2 subsequently spread rapidly, and was common in dogs worldwide by 1980. Although its origin remains uncertain, it is similar to mink enteritis virus and feline panleukopenia virus (FPV), and possibly arose from a mutation of FPV or a third closely related virus (Pollock 1984).

The first evidence of exposure to CPV-2 among wolves and coyotes in North America was apparently in 1978–1979, based on retrospective serological studies (Barker et al. 1983, Thomas et al. 1984, Mech et al. 1986), although Goyal et al. (1986) provided evidence of CPV exposure in wolves in Minnesota as early as 1975. During 1978–1983, Mech et al. (1986) reported an increased seroprevalence in wolves in Minnesota that paralleled results from surveys in coyotes in Texas, Utah, and Idaho (Thomas et al. 1984). Among coyotes, seroprevalence increased rapidly from 0% in 1979 to ≥ 50% in 1980. Positive hemagglutination-inhibition (HI) titers predominated through 1983, when data were last reported, suggesting the disease was enzootic in coyotes (Thomas et al. 1984). The prevalence of positive HI titers among wolves in Minnesota reached 65% in 1980, and ranged between 36% and 44% during 1981–1983 (Mech et al. 1986). In a separate study in north-central Minnesota, CPV-2 antibody was not found in 11 wolves sampled in 1977; but during 1981–84, 15 (26%) of 57 wolves were seropositive (T.K. Fuller, Univ. of Massachusetts, pers. commun.).

In south-central Alaska, the first positive serum neutralizing titer in wolves was reported in 1980, and during the next two years 50% of 18 wolves sampled had positive titers to CPV-2 (Zarnke and Ballard 1987). Similarly, initial CPV-2 titers among wolves on the Kenai Peninsula in south-central Alaska were recorded in 1979 (Bailey et al. this volume). R.P. Thiel (Wisconsin Dept. of Natural Resources, pers. commun.) found positive HA titers to CPV-2 in 67% of 24 wolves from Wisconsin. He also recovered the remains of four wolves from Wisconsin that appeared to have died from disease and parasitism; two of these wolves were previously seropositive to CPV-2, although CPV-2 was not considered a primary cause of death.

There are no published reports of mortality or clinical illness from CPV-2 among free-ranging wolves, although losses of captive wolves have been high, as in other canids. In 1983, CPV-2 claimed 11 of 12 pups and yearlings in a captive wolf colony in Minnesota (Mech and Fritts 1987). J. Zuba (Univ. of Wisconsin College of Veterinary Medicine, pers. commun.) conducted the only experimental study of the effects of CPV-2 on wolves. Results of this study were similar to those conducted on dogs. Wolves that were challenged with the virus seroconverted and thereafter had positive titers to CPV-2; about 30% of these animals showed clinical signs of disease, and about 10% of the animals would probably have died without supportive care.

CPV-2 became the focal point of concern in the late 1980's as the wolf population in Isle Royale National Park (Michigan) declined to an all-time low level (R.O. Peterson, unpubl. data). A spectacular crash occurred in 1980–1982 when this island population dropped from a maximum of 50 to 14 wolves, cumulatively including the deaths of more than 52 individuals. All nine wolf pups known to be alive in 1981 died before midwinter surveys began, coincident with an outbreak of CPV-2 among dogs in Houghton, Michigan, the mainland departure point for visitors to the island. While the presence of CPV-2 on Isle Royale was confirmed by the presence of positive titers in several wolves in the late 1980's (N.J. Thomas, Natl. Wildl. Health Res. Center, pers. commun.), the link between high mortality in the early 1980's and CPV remains circumstantial.

Oral papillomatosis

Oral papillomatosis was reported in two wolves (and 10 coyotes) from Alberta during 1971–1976 (Samuel et al. 1978). These two wolf pups were found dead together near a poisoned bait center. This viral disease resulted in mild infection of the lips with multiple (<20) tumors in the wolf pups, although lesions in the coyotes ranged from mild to severe (Fig. 1).

Debilitation or mortality from oral papillomatosis has not been reported in free-ranging canids. The disease probably does not cause direct mortality, but may alter behavior or feeding, as suggested for coyotes (Trainer et al. 1968), result in secondary infections, or be associated with other debili-

Fig. 1. *Canine oral papillomatosis in a coyote from Alberta. Photo: M.J. Pybus.*

tating diseases such as mange. Spontaneous recovery with long-lasting immunity has been suggested in coyotes (Trainer et al. 1968).

Bacterial and Fungal Diseases

Brucellosis

Brucellosis is a contagious disease caused by the bacterium *Brucella* spp., including up to five recognized strains. The disease primarily affects ruminants, often resulting in abortion, orchitis, or other reproductive disorders.

In Alaska, Neiland (1975) reported on seroprevalence to *Brucella* spp. in wolves and other carnivores that were associated with infected caribou (*Rangifer tarandus*) in the Arctic herd. A 45% (10/22) prevalence of agglutinating titers among adult wolves contrasted to the 9% (1/11) prevalence among red foxes and 7% among sled dogs, presumably a result of transmission from consumption of caribou infected with *B. suis* biovar 4. Neiland (1975) found no serologic reactors among 98 wolves tested from the Porcupine caribou herd range and on St. Lawrence Island. Zarnke and Ballard (1987) reported 1% (1/67) antibody prevalence among wolves in south-central Alaska. They attributed this low rate to the relatively low infection rate in caribou of the Nelchina herd (less than 5% seropositive) and the increased use of moose (*Alces alces*), a species rarely infected with brucellosis. Pinigan and Zabrodin (1970) found 11% (12/110) of wolves exposed to brucellosis, presumably *B. suis* biovar 4, in Siberian reindeer (*Rangifer tarandus*) ranges. In Alberta, 31% (4/13) of wolves examined in Wood Buffalo National Park were infected with *Brucella abortus* biotype 1 (S.V. Tessaro, Agriculture Canada, pers. commun.); however, titers were not detected in three wolves collected 80 km south of the park (Zarnke and Yuill 1981).

The effects of *Brucella* infection in wolves under natural conditions is unknown. Neiland and Miller (1981) experimentally infected two gravid wolves with *B. suis* biovar 4; although clinical disease was not observed in these wolves, four of six pups in one litter were born dead. The surviving pups were killed by the bitch within 24 hours of birth. Although brucellosis was not diagnosed as the cause of death among the pups, *B. suis* biovar 4 was isolated from the liver of each of the seven pups and from the spleen of five. *Brucella suis* biovar 4 was also isolated from a wide variety of organs of both bitches, including the liver, spleen, mam-

mary glands, and lymph nodes; the uterine horns were infected in one. Whether these deaths could be attributed indirectly to brucellosis is not known, however, consumption of aborted fetuses and placentae by infected dogs is common (Carmichael and Kenney 1970). Neiland and Miller (1981) concluded that reproductive failure was a "probable, but essentially unproven, consequence of ill-timed infections" in wolves.

Leptospirosis

Leptospirosis is a bacterial infection caused by the genus *Leptospira*, and includes 170 known species, or serovarieties. Evidence for infection in wolves is limited. Zarnke and Ballard (1987) found detectable antibodies in only 1/82 (1%) wolves sampled in south-central Alaska. However, Khan et al. (1991) found serologic evidence of infection in 52 (11.4%) of 457 wolves tested from northern Minnesota. They identified nine species: *L. grippotyphosa* (5.3%), *L. bratislava* (3.9%), *L. autumnalis* (3.3%), *L. canicola* (2.8%), *L. pomona* (1.5%), *L. pyrogenes* (1.5%), *L. ballum* (0.7%), *L. copenhageni* (0.7%), and *L. hardjo* (0.4%).

Sources of infection in wild mammals are from infective urine, and among carnivores, through the food chain (Reilly et al. 1970). Interspecies transmission to wolves is possible through predation and scavenging or intraspecies by contact with urine, such as through scent marking. In northern Minnesota, seroprevalence to one or more species was 2.6 times greater in wolves near farming areas (20.1%) than in wolves from nonfarming areas (7.7%) (Khan et al. 1991). This difference is possibly due to increased contact with infected livestock and contaminated livestock waste. Leptospirosis is endemic in bovine, porcine, and equine populations in Minnesota (Khan et al. 1991). However, *L. grippotyphosa*, the most prevalent species in wolves (5.3%), was also found in 89 (27.1%) of 328 moose (Diesch et al. 1972), yet was the least common species in domestic livestock in Minnesota (Khan et al. 1991).

Leptospirosis ranges from an inapparent to fatal disease, depending on host and serovariety. Clinical disease or population effects of leptospirosis in wild canids have not been reported. In domestic dogs, disease conditions from mild unapparent to severe are caused primarily by *L. icterohaemorrhagiae* and *L. canicola* (Alston et al. 1958).

Limited information does not suggest that leptospirosis is important in wolves; however, this disease may warrant concern where desired wolf reintroduction or recolonization sites include areas of enzootic leptospirosis among prey or other carnivore species and where wolves may act as a reservoir and source of infection for wild and domestic animals.

Lyme disease

Lyme disease (borreliosis), caused by the bacterium *Borrelia burgdorferi*, affects humans, horses, and dogs. The disease was first recognized in New England in 1975, and possibly as early as 1969 in Wisconsin, and has since been reported with increasing frequency in at least 43 states and in eastern Canada. Infection usually results from the bite of infected ticks, primarily *Ixodes dammini*. White-tailed deer (*Odocoileus virginianus*) serve as hosts for adult *I. dammini* ticks while small mammals, primarily white-footed mice (*Peromyscus leucopus*) and eastern chipmunks (*Tamias striatus*), are hosts for immature ticks. Although these hosts become infected with *B. burgdorferi*, they do not appear to show clinical signs of disease. Burgess and Windberg (1989) provided evidence of transplacental transmission of *B. burgdorferi* infection in free-ranging coyotes, and J.M. Gustafson (Univ. of Wisconsin College of Veterinary Medicine, pers. commun.) reported transplacental transmission in natural- and laboratory-infected ranch-raised foxes. Contact transmission has also been reported in dogs and ranch-raised foxes (Burgess 1986; J.M. Gustafson, pers. commun.).

Evidence that wild wolves become infected with Lyme disease is limited to a serosurvey of trapped wolves in Wisconsin and Minnesota (Kazmierczak et al. 1988). Two of 78 wolves were positive with the indirect fluorescent antibody test; one had a titer indicative of active infection, whereas a low titer in the other wolf suggested either an early or late stage of infection or transient exposure without actual infection.

Although clinical Lyme disease has not been found in wild wolves, Kazmierczak et al. (1988) demonstrated potential susceptibility through intravenous inoculation of one wolf with *B. burgdorferi* in the laboratory. Lymphadenopathy (disease of the lymph nodes) was observed, but other manifestations of disease were not present. Subcutaneous inoculation of a different wolf and ingestion of suckling white-footed mice inoculated with the bacterium by two others did not result in infection. However, in dogs, Lyme disease is characterized by arthritis, arthralgia, fever, and lymphadenitis (Lissman et al. 1984, Kornblatt et al. 1985). Abortion and fetal mortality have been reported in infected humans and horses (Schlesinger et al. 1985, Burgess et al. 1989). Effects on reproduction in infected wolves are not known.

Tularemia

Tularemia, caused by the bacterium *Francisella tularensis*, has not been reported in wolves, although coyotes and red, gray, and kit (*Vulpes macrotis*) foxes are susceptible (summarized in Bell and Reilly 1981). Signs of tularemia in red foxes include anorexia, diarrhea, and noisy, labored breathing. Pathologic changes in red and gray foxes include enlargement of lymph nodes, liver, and spleen; necrosis in the liver and spleen; and congested tubercle-like areas or diffuse consolidation in the lungs.

Zarnke and Ballard (1987) reported a seroprevalence to tularemia of 25% in wolves from Alaska and speculated that most healthy adults probably recover from the disease. Transmission of tularemia to carnivores is most likely from infected lagomorph or rodent prey.

Bovine tuberculosis

Bovine tuberculosis, caused by the bacterium *Mycobacterium bovis,* is primarily a disease of cattle and other ungulates (Thoen and Hines 1981, Tessaro 1986). Other species, including carnivores, may become infected, but infections are probably limited to individual or small local populations closely associated with ungulates.

Carbyn (1982b) reported the occurrence of bovine tuberculosis in wolves from Riding Mountain National Park, Manitoba; this is the only published account of the disease in wild wolves. Among 21 wolves radio-collared during 1975–1979, two of 14 known deaths were attributed to tuberculosis, one isolate being identified as *M. bovis*. Both deaths occurred in pups, presumably litter mates, in an emaciated condition. The source of infection was not identified. Transmission to these siblings could have been from infected prey or carrion, an infected bitch, or contaminated soil. *Mycobacterium bovis* can survive in the environment a few weeks, although some species of mycobacteriae can remain viable in soil for four years or more (Thoen and Hines 1981). Lesions observed were characteristic of tuberculosis in domestic animals and included nodules in the lung and liver and enlargement of intestinal lymph nodes. Carbyn (1982b) attributed the decline in the wolf population (from 120 in 1975 to 63 in 1978) partly to disease, including tuberculosis.

In Wood Buffalo National Park, where bovine tuberculosis is enzootic in bison (*Bison bison*), there was no evidence of infection in 13 wolves examined as part of an epidemiological study of tuberculosis (S.V. Tessaro, Agriculture Canada, pers. commun.).

Blastomycosis

Thiel et al. (1987) reported a fatal case of the fungal disease blastomycosis (*Blastomyces dermatitidis*) in a wolf in Minnesota. Blastomycosis is enzootic in Minnesota (Schlosser 1980) and Wisconsin (Sarosi et al. 1979, McDonough and Kuzma 1980) and is a problem in dogs in these states (Archer 1985). The fungus is probably transmitted from point sources in the environment; the disease is not contagious. The limited distribution of blastomycosis suggests that its potential effects are limited to wolf populations in Wisconsin and Minnesota.

Helminths

At least 24 species of nematodes (roundworms), 21 species of cestodes (tapeworms), nine species of trematodes (flukes), and three species of acanthocephala (spiny-headed worms) have been reported from gray wolves (Mech 1970). In addition to general survey reports and individual species reports, there have been rigorous community analyses of the helminth fauna of wolves throughout North America. Custer and Pence (1981a) used similarity indices and multivariate analyses to compare seven parasite surveys conducted from northern Alaska and Canada to the southern United States.

Wolves have a characteristic helminth fauna and high index of similarity throughout much of their range (Holmes and Podesta 1968, Custer and Pence 1981a). Cestode species provide the most predictable element of parasite communities in wolves; in particular, taeniid cestodes (Freeman et al. 1961, Holmes and Podesta 1968, Custer and Pence 1981b), many of which use vertebrate intermediate hosts to complete the life cycle.

Custer and Pence (1981a) defined two regional clusters of helminth communities in gray wolves: northern regions (Alaska, Yukon, and Northwest Territories), and southern regions (Minnesota, Manitoba, and Alberta). Regional differences in species composition largely reflect differences in wolf diets and associated parasites of prey species. Wolf populations in the far north (characterized by a common occurrence of *Taenia krabbei* and, less frequent, *T. hydatigena* and *Echinococcus granulosus*) feed almost exclusively on cervids, particularly moose and caribou (Mech 1970, Choquette et al. 1973). Southern populations (characterized by common *T. hydatigena*, moderate *E. granulosus*, and low *T. krabbei* occurrence) rely more heavily on white-tailed deer (Thompson 1952, Stenlund 1955, Pimlott et al. 1969, Mech 1970) and beaver (*Castor canadensis*) (Pimlott et al. 1969, Peterson 1977, Shelton and Peterson 1983).

Helminth parasites of wolves often have limited pathogenicity and thus have minimal effect in regulating wolf populations. This likely reflects the predominance of tapeworm infections since the worms feed on nutrients absorbed from the gut contents rather than from the host itself. Tapeworms attach to the intestinal wall simply as a holdfast (to avoid being swept away) and not as a means of damaging the gut wall to feed on blood or tissues. In contrast, there is potential for damage to individual wolves from some species of nematodes and trematodes.

Dog heartworm

The dog heartworm, *Dirofilaria immitis*, is found in the heart and pulmonary arteries of a variety of hosts (Fig. 2), particularly domestic dogs. Mosquitoes are the main vectors of transmission. Pathologic changes and death from dog heartworm have been reported in gray wolves held in zoos in enzootic areas (Hartley 1938, Coffin 1944, Pratt et al. 1981). Clinical pathology in wolves includes detectable heart murmurs and pulse deficits; gross pathology includes cardiac enlargement and chronic passive congestion (Pratt et al. 1981). Preventive medication is recommended when captive wolves are maintained in heartworm enzootic areas. Canine heartworm may have been a significant factor in the decline of red wolves (*Canis rufus*) in the southeastern U.S. (McCarley and Carley 1979). Mech and Fritts (1987) reported *D. immitis* in free-ranging wolves in Minnesota and expressed concern over the possible effects on wolf populations in Minnesota and Wisconsin.

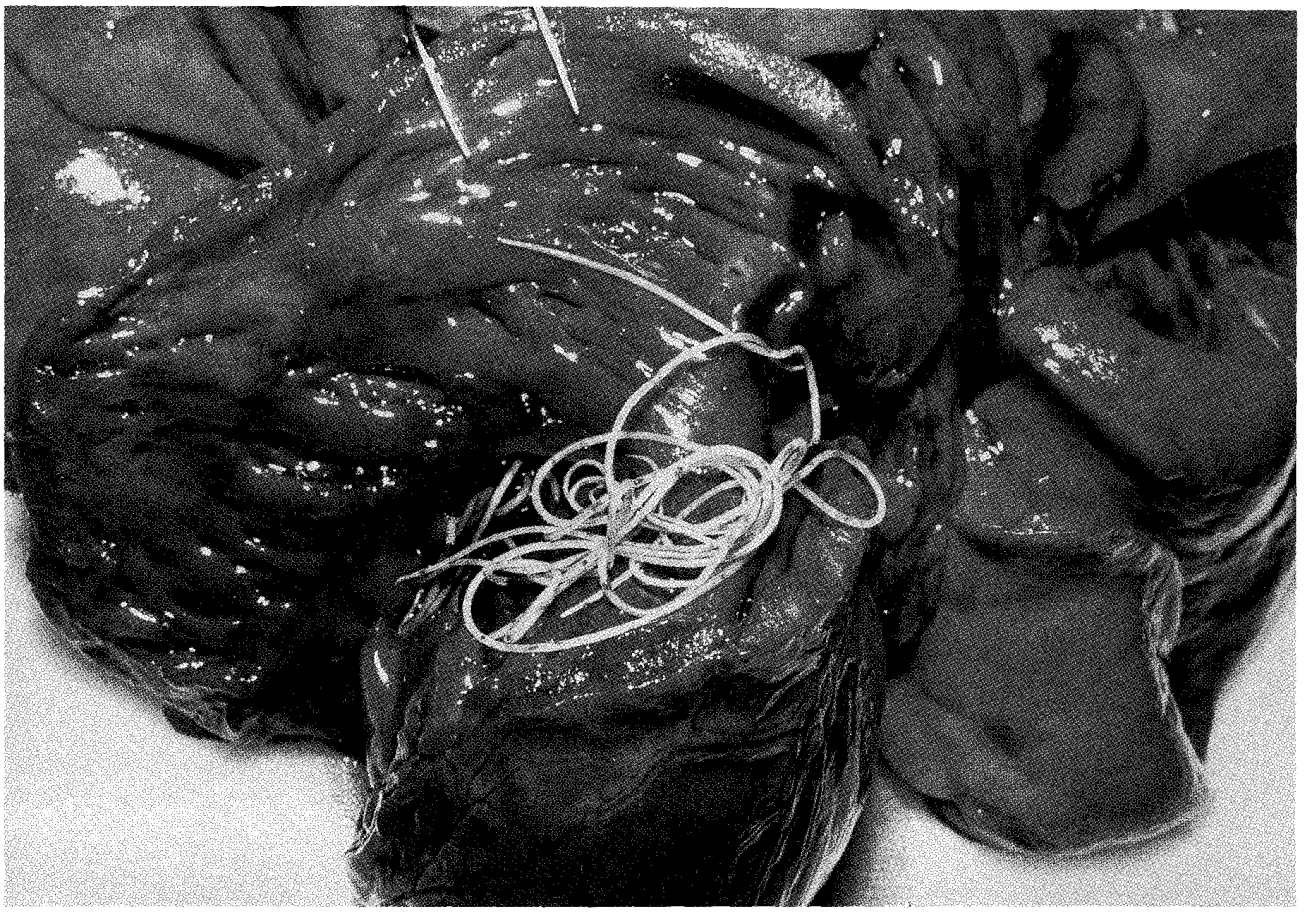

Fig. 2. *Heartworms (**Dirofilaria immitis**) in a red wolf from Alligator National Wildlife Refuge, North Carolina. Photo by N.J. Thomas.*

Dog Hookworm

The dog hookworm, *Ancylostoma caninum*, is a large blood-feeding nematode which attaches to and abrades the intestinal wall. Infections in dogs have been associated with anaemia, emaciation, diarrhea, and death. In free-ranging canids, mortality of infected red wolves (McCarley and Carley 1979, Custer and Pence 1981a) and coyotes (Mitchell and Beasom 1974) is suspected. Although this parasite has not been reported in gray wolves, it may be a threat where it is enzootic in other canids. A different species of hookworm, *Uncinaria stenocephala*, has been reported in gray wolves but its pathogenicity has not been assessed.

Liver fluke

Metorchis conjunctus, a trematode found in the gall bladder and bile duct of a variety of fish-eating mammals, has been implicated as a potential pathogen of wolves. It was found in one of 98 wolves from Alberta (Holmes and Podesta 1968) and seven of 211 wolves from Saskatchewan (Wobeser et al. 1983). In Saskatchewan, five of seven cases were from a population of wolves known to consume fish. No pathologic change was seen in the wolf from Alberta; however, thickened nodules or cord-like swellings (greatly dilated bile ducts) were seen throughout the liver of infected wolves from Saskatchewan; and in two cases, infections also were associated with extensive damage to the pancreas. Wobeser et al. (1983) concluded that damage to the pancreas could affect endocrine or exocrine function but could not determine whether health of infected wolves was impaired. Population regulatory effects of this fluke are not known, but, if they occur, would be restricted to local populations that consume fish.

Hydatid tapeworm

Although the hydatid tapeworm, *Echinococcus granulosus*, does not directly cause mortality in wolf populations, Messier et al. (1989) considered it an integral part of the moose-wolf population dynamics in southwestern Quebec. These authors documented a direct relation between prevalence of hydatid cysts in moose, density of wolves, and rate of wolf predation on moose. They proposed the following regulatory mechanism: as density of wolves increases, sites used extensively by wolves are contaminated with large numbers of *E. granulosus* eggs in wolf feces. The prevalence and intensity

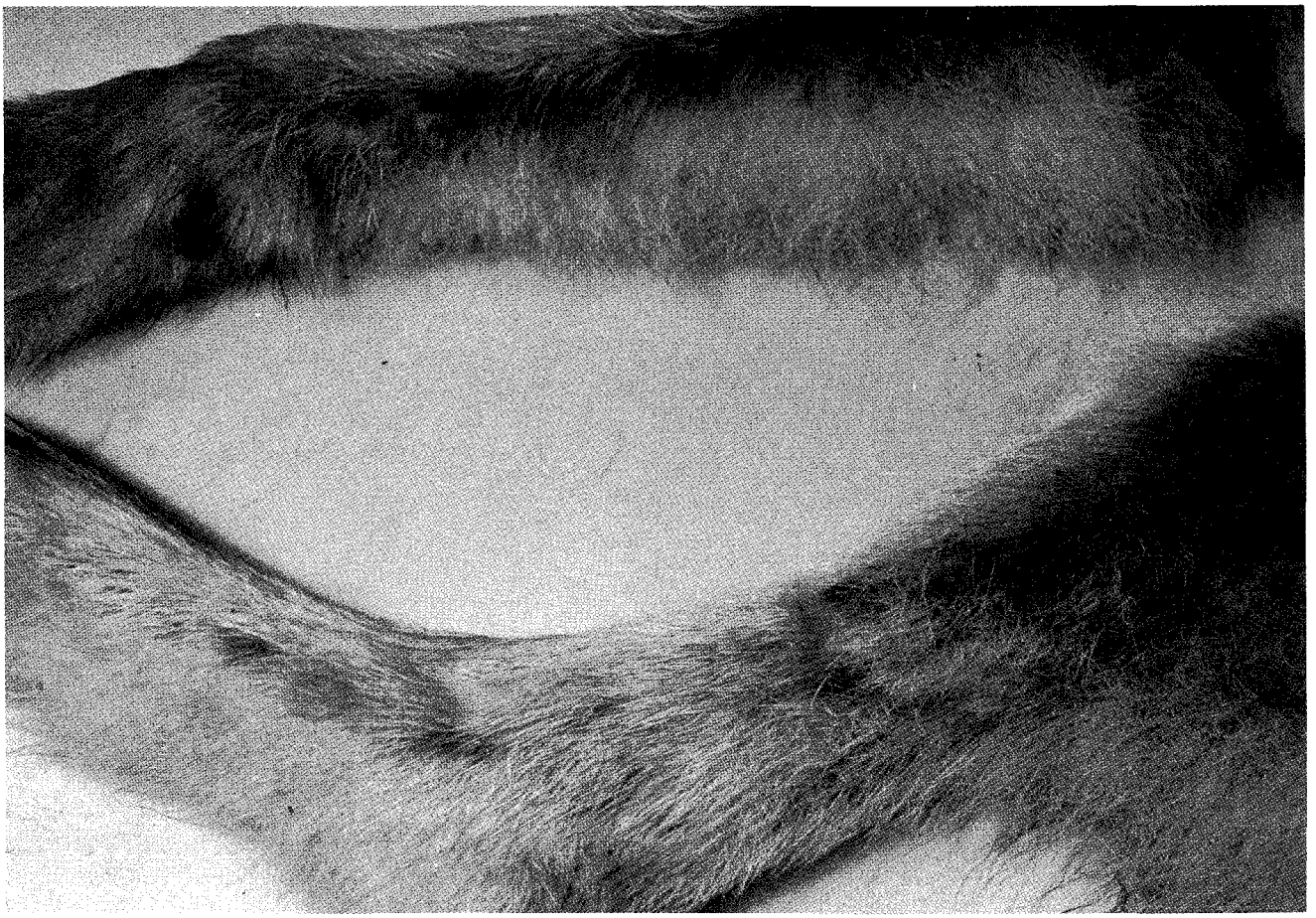

Fig. 3. *Alopecia resulting from louse (**Trichodectes canis**) infestation in a gray wolf from Minnesota. Photo by J. Runningen.*

of hydatid cysts increase in moose populations using these areas. Since most cysts occur in pulmonary tissue (Sweatman 1952, Addison et al. 1979), moose with large numbers of cysts are to likely suffer decreased stamina (Cowan 1951, Mech 1966a, Rau and Caron 1979) and increased physical impairment (Messier et al. 1989). Heavily infected moose may display behavioral changes detected by wolves (Mech 1970) and be selected as prey. Thus, the parasite enhances the regulatory effect of wolf predation on moose (Messier and Crete 1985) and affects wolf population dynamics through increased prey susceptibility.

Ectoparasites

There are few reports of ectoparasites on gray wolves. Fleas (*Pulex simulans*, *Ctenocephalides canis*) (Skuratowicz 1981, Hristovski and Beliceska 1982), ticks (*Amblyomma americanum*, *A. maculatum*, *Dermacentor albipictus*, *D. variabilis*, *Ixodes* spp.) (Pence and Custer 1981, Archer et al. 1986), and deer fly (*Lipoptena cervi*) (Itamies 1979) occur, but infestations appear rare. The major ectoparasites on wolves are lice and mites.

Lice

The dog louse (*Trichodectes canis*) has been reported recently on free-ranging gray wolves throughout most of their range in North America. Infestations on dogs in North America are common and likely are the source of initial infestations in wild canids. Lice are transmitted by direct contact between infested and uninfested individuals. They transfer readily from females to pups.

Louse infestations on wolves involve varying degrees of alopecia (hair loss) (Fig. 3). Guard hairs often are missing or broken and underfur is matted (Schwartz et al. 1983, Mech et al. 1985). The matting of the fur tends to distinguish louse infestations from sarcoptic mange (see below). Damage to the hair is self-inflicted and reflects attempts to remove the lice by biting, chewing, and scratching. Damage is most often seen on the shoulders and groin; but, in severely affected wolves, only the head, legs, and tail remain undamaged. In addition, a "mousy" smell often is associated with infested wolves. Pups appear to be affected more frequently and severely than adults.

There is little evidence that *T. canis* directly affects the dynamics of wolf populations (Schwartz et al. 1983, Mech et al. 1985). Adult wolves with severe alopecia and secon-

Fig. 4. *Sarcoptic mange in a gray wolf from Alberta. Photo by J.R. Gunson.*

dary inflammation and bacterial infections were reportedly in good body condition. Severe infestations may contribute to reduced survival of individual pups (Schwartz et al. 1983), but this has not been confirmed (Mech et al. 1985).

In 1981 and 1982, *T. canis* was reported in five of 20 wolf packs on the Kenai Peninsula, Alaska. Wildlife managers were concerned that lice would spread to other packs in Alaska and northern Canada. Thus, federal and state officials treated infested wolves with ivermectin administered by intramuscular injection or in treated baits (Taylor and Spraker 1983). The intensive treatment program continued for two to three years. Currently, the dog louse occurs on wolves throughout the Kenai Peninsula, but has not been reported elsewhere in Alaska (R.L. Zarnke and T. Spraker, Alaska Dept. of Fish and Game, pers. commun.).

Mites (mange)

Sarcoptic mange is the most conspicuous and probably most significant ectoparasite of wolves. The mite, *Sarcoptes scabei*, is distributed worldwide, exhibits little host specificity, and transfers readily among a variety of host species (Sweatman 1971). In North America, it is common on red foxes but also occurs on coyotes and wolves throughout their range. There is a long history of mange or "mange-like" conditions in free-ranging canids in North America. Pike (1892:53) concluded that a disease resembling mange was responsible for the death of numerous hairless wolves throughout northern regions. As early as 1909, sarcoptic mange was introduced into Montana on large numbers of experimentally infested coyotes and wolves in an attempt to control free-ranging canids (Knowles 1909:130, 1914:229-230). This experiment may have been the source of mange on wild canids in western Canada (Green 1951). Currently, mange is enzootic in western Canada, and its effects on pup survival may be significant in cyclic population fluctuations in wolves throughout the region (Todd et al. 1981).

Sarcoptic mites cause extensive irritation and damage as they burrow into skin and tunnel within the epidermis. The life cycle is short and new generations of mites can appear every 14 days (Sweatman 1971). Mites are transferred to new hosts by direct contact with infested animals or by using rubbing posts contaminated with mites.

Wolves with mange usually have severe hair loss with relatively little exudate or crusting (Fig. 4). Severe infestations often involve extensive alopecia, crusted lesions, and thickened, slate-gray skin over much of the body. Heavily

infested wolves can have lower weight and fat deposits than uninfested animals (Todd et al. 1981). Loss of condition is more marked in pups than in adults. Behavioral changes relating to food habits have been documented in infested coyotes (Todd et al. 1981). Similar behavior of infested wolves was suspected but not verified.

Based largely on circumstantial evidence, several researchers believe mange is an important regulating factor in wild canid populations (Pike 1892:53, Murie 1944, Cowan 1951, Green 1951, Todd et al. 1981). During a 10-year period in Alberta, mange was present each year, but the prevalence differed annually and locally. The number of cases increased when wolf densities increased, and the number of surviving pups decreased as the prevalence of mange increased (Todd et al. 1981).

Discussion

Diseases and parasites must affect reproduction, mortality, immigration, or emigration (dispersal) to be important in population dynamics of wolves. Effects need not result in death, but can include sublethal effects on physiological and homeostatic processes, thriftiness, reproduction, and behavior, which in turn can affect wolf population dynamics.

Assessing direct and indirect influences of diseases on wolf populations is difficult for a variety of reasons:

1) Wolves may die directly from disease or parasites, but the probability of locating carcasses is remote unless individual survival is monitored intensively (e.g., by radiotelemetry). Scavenging, decomposition, and freezing can render carcasses and tissues unsuitable for necropsy, histopathology, and supporting diagnostic tests.
2) Important population processes such as fetal and neonatal survival are difficult or impossible to monitor in free-ranging wolves.
3) Sublethal effects of disease are extremely difficult to diagnose and document in wild populations. Clinical or pathological evidence that a disease is contributing or predisposing to another cause of death is rarely available and, if available, is usually speculative.
4) Multiple infectious and parasitic agents are frequently found at necropsy. Evaluating the significance of multiple agents and their additive and synergistic effects is difficult and often speculative. For example, Appel (1988) reported that dogs suffered high mortality from dual infections of CPV-2 and canine corona virus even though neither virus by itself was highly virulent.
5) Contributing factors, such as food shortage leading to nutritional stress, may combine with disease factors to increase the significance of otherwise innocuous or sublethal infections.
6) Interpretation of disease prevalence in a population based on serological data can be misleading because seropositive animals represent only survivors of exposure rather than incidence or prevalence of disease.
7) Experimental studies of diseases in captive and free-ranging wolves employing adequate controls are lacking.
8) Long-term studies of wolf populations are few, yet these will probably provide the most important data by which population processes are elucidated.
9) Where population density alone is estimated on an annual basis, increased reproductive success may compensate for high mortality rates, hiding important demographic responses to disease. For instance, annual mortality rates of > 35% were required to measurably reduce wolf density (Gasaway et al. 1983, Keith 1983, Peterson et al. 1984, Ballard et al. 1987, Fuller 1989). Otherwise, increased reproduction or reduced dispersal may compensate for increased mortality.

Direct and circumstantial field evidence and extrapolation from studies in captive wolves and other canids suggest that diseases and parasites affect population dynamics through direct and indirect means. Mortality of wolves in the wild has been documented for rabies, canine distemper, parvovirus, blastomycosis, tuberculosis, and mange; in some instances, epizootics were associated with population declines (Davis et al. 1980, Carbyn 1982b, W.B. Ballard, unpubl. data, R.O. Peterson, unpubl. data). Other parasites and diseases such as canine heartworm, hookworm, and infectious canine hepatitis that can be fatal to other canids are also present in wild wolf populations, but there is little or no evidence that these diseases directly cause mortality or affect gray wolf populations.

Circumstantial evidence further suggests that sublethal infections of some parasites and diseases also affect wolf populations. Infections that can debilitate or alter behavior in other canids (Lyme disease, oral papillomatosis, sarcoptic mange) may have similar effects in wolves, but extrapolation between species and between captive and wild conditions should be made with caution. Likewise, inferences from other canids on potential effects on reproduction from diseases such as brucellosis and Lyme disease also require caution.

Despite the evidence of parasites and diseases affecting some wolf populations, the role of disease in limiting wolf populations remains unknown. In other canid populations, relations between population density and some diseases such as sarcoptic mange (Todd et al. 1981), rabies (Debbie 1991, Fekadu 1991), and canine distemper (Trainer and Knowlton 1968) have been suggested.

Management Considerations

Management of the wolf in North America has relied traditionally on information from research and monitoring of population size and structure, predation and diets, social organization and structure, home ranges and movements, recruitment, and mortality rates. The influence of diseases on these population characteristics has not been fully recog-

nized by some agencies, yet limited information summarized here suggests that disease may be an important factor in some cases. Knowledge of diseases in a population and how they influence wolf populations may be important to management, whether they are enzootic (e.g., canine distemper virus, infectious canine hepatitis) or sporadically introduced from other sources (dog louse, rabies, CPV-2). The role of the wolf as reservoir or host of some diseases may also have important implications for public health and the health of domestic animals and other wildlife.

Man's impact on the environment may play a role in the composition of diseases in wolves and their effects on wolf populations. For example, increased contact between domestic pets and livestock may result in the establishment of diseases not present or not important in historic times, such as CPV-2. Fragmentation of wolf habitat may result in more isolated wolf populations that may be more severely affected by new or existing diseases, particularly in areas of reintroduction into former wolf range.

Current efforts to reintroduce the wolf to its former range will be successful only if individuals survive and reproduce. Disease potential in areas of reintroduction should be one of the many considerations in planning such programs (Johnson this volume). The presence and risk of diseases that can be transmitted to wolves at relocation sites, particularly from other wild canids and domestic and feral dogs should be evaluated; special attention should be paid to diseases that are not present in the originating wolf population. Prophylaxis (prevention) for certain diseases has been recommended for individual relocated wolves, including vaccination for rabies, canine distemper, CPV-2, leptospirosis, and ICH, and those diseases of which we know little in wolves (influenza, parainfluenza, and corona virus [Albert et al. 1987]). However, there are little data on the efficacy or safety of domestic animal vaccines in wildlife (Fowler 1978). Although some vaccinations may provide at least some degree of protection, little is known about the duration of protection for standard canine vaccines in wolves, and progeny would not be protected beyond the duration of maternal antibody. Modified live vaccines (MLV) have been generally proven to be more effective than killed vaccines. Although captive wolves are frequently vaccinated with MLVs, these vaccines can produce active disease, particularly in species for which the vaccine was not developed (Fowler 1978).

Diseases should also be considered in selecting areas for wolves to be captured for relocation. Introduction of diseases from originating populations to the release site could jeopardize other wildlife and domestic animals and the success of the reintroduction. Health assessment for wolves to be reintroduced should include testing for specific parasites and diseases, and appropriate treatment and prophylaxis (Albert et al. 1987).

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